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Science, (Oct., 1999) Vol. 41, No. 5, pp. 563-574.

ANTI- ***STREPTOCOCCUS*** - ***MUTANS*** ***IGY*** . TAGUCHI T; HIRASAWA M; ASAKA H; HONDA M; NIIHO K; OTAKE S. NIHON UNIV. SCH. DENT. MATSUDO, JPN.. JOINT MEETING OF THE 70TH GENERAL MEETING OF THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH (IADR), 40TH ANNUAL MEETING OF THE BRITISH DIVISION OF THE IADR, 1992 ANNUAL MEETING OF THE CONTINENTAL EUROPEAN DIVISION OF THE IADR, 8TH ANNUAL MEETING OF THE IRISH DIVISION OF THE IADR, AND THE 75TH ANNUAL MEETING OF THE SCANDINAVIAN ASSOCIATION FOR DENTAL RESEARCH, GLASGOW, SCOTLAND, UK, JULY 1-4, 1992. **J DENT RES. (1992) 71 (SPEC ISSUE), 650.** CODEN: JDREAF. ISSN: 0022-0345. Language: English.

Nippon Nogeikagaku Kaishi, (1993) Vol. 67, No. 10, pp. 1437-1439. ISSN: 0002-1407. Language: Japanese.

MICROBIOLOGY AND IMMUNOLOGY, (1978) 22 (6): 301-14.

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1075 Anticaries Effect of Tea Catechins and anti-*S.mutans* IgY. T.TAGUCHI*, M.HIRASAWA, H.ASAKA, M.HONDA[1], K.NIIHO[1] and S.OTAKE (Nihon Univ. School of Dent. at Matsudo, [1]Kowa Display Tokyo, JAPAN).

Our previous study show that feeding of Sunphenon™ (tea catechins) and anti-*Streptococcus mutans* hen egg yolk powder (HEY) results in reduction of caries development in rats. The incidence of dental caries in rats infected with *S.mutans* JC-2(*c*) and fed with a modified diet 2000 containing 56% chocolate (approximately 25% sucrose) which supplemented with 0.1% Sunphenon™ was significantly lower ($p<0.01$) than similarly infected rats given a 56% chocolate diet only. In the further experiment, all rats were fed with modified diet 2000, in which 56% sucrose in diet 2000 was replaced by 36% sucrose and 20% HEY powder, which contained immunized and control powder and/or supplemented with 0.05% Sunphenon™ in drinking water. Four groups of rats were fed with diet containing four different ratios of anti-*S.mutans* immunized yolk and control yolk powder, as follows : 100/0 for group A ; 33/67 for group B; 10/90 for group C ; 0/100 for group D. Further, another rats of group A-D received drinking water of which contained 0.05% Sunphenon™. Rats infected with *S.mutans* and fed with modified diet 2000 containing HEY and Sunphenon™ showed lower caries score ($p<0.05$) than similarly infected rats given the diet containing HEY singly. These data suggest that addition of Sunphenon™ to food reduces its caries potential, and the inhibitory effect of caries development between immune-HEY and tea catechins may react to different site.

1076 Passive Immunization with Anti-*S.sobrinus* IgY against Dental Caries in Rats. Y.NISHIHARA*, M.HIRASAWA, M.FUKUMOTO, T.KUROKI, H.HATTA[1] and S.OTAKE (Nihon Univ. School of Dent. at Matsudo, [1]Taiyo Kagaku Co.,Ltd.,Mie,JAPAN).

Our previous study show that feeding anti-*S.mutans* MT8148 (*c*) immunoglobulin egg yolk (IgY) result in reduction of caries development in rats. In the present study, the preparation of IgY against *S.sobrinus* 6715 (*g*) that was cultivated in medium containing either in absence (suc$^-$) or presence (suc$^+$) of sucrose.The summary of results were described as follows; 1. When cross reactivity was examined by ELISA, this anti-*S.sobrinus* IgY (suc$^-$ or suc$^+$) specifically reacted with serotypes *d*, *g* and *h* cells, which were obtained from medium without or with sucrose. On the other hand, anti-*S.sobrinus* IgY (suc$^+$) cross reacted with *a*, *c*, *d*, *e*, *f*, *g* and *h* serotypes of mutans streptococci prepared from media containing 5% sucrose. 2. Rats infected with *S.sobrinus* 6715(*g*), *S.sobrinus* OMZ65(*g*) or *S.sobrinus* OMZ176(*d*) and fed with cariogenic diet containing anti-*S.sobrinus* 6715 (suc$^-$ or suc$^+$) egg yolk had significant lower caries scores than fed with a diet containing non-immunized egg yolk. Rats fed with cariogenic diet containing anti-*S.sobrinus* (suc$^+$) egg yolk and infected with serotype *c* strain had significantly lower caries score than the identically challenged non-immunized egg yolk group. However, rats fed with diet containing anti-*S.sobrinus* (suc$^-$) egg yolk and infected with serotype *c* strain MT8148 showed no difference at a significant level.

1077 Avidity of antibodies to *Porphyromonas gingivalis* and periodontal status. J. Mooney*, E. Adonogianaki and D.F. Kinane (Oral Medicine & Pathology, Glasgow University, UK).

This study investigated the titre and avidity of IgG, IgM and IgA serum antibodies to *P. gingivalis* in patients with periodontal disease and healthy subjects, cross-sectionally and longitudinally. Thirty-one subjects were studied, eleven healthy controls and two periodontitis groups comprising:- a) fourteen subjects who went on to demonstrate attachment loss in at least one site over a three month period (AL), and b) six subjects who did not exhibit attachment loss (NAL) (detected using the Florida probe and the tolerance method). The titre and avidity of IgG, IgM and IgA antibodies to *P. gingivalis* in the sera of these subjects was assayed by ELISA (Ebersole *et al*, *J Periodont Res* 15:621-632,1980). Conversion to ELISA units (EU) was performed using the method of Gmur *et al* (*Infect Immun* 52:768-776,1986) and antibody avidity was expressed as molarity (M). The periodontitis subjects generally showed higher IgG (2390EU median, p=0.0053) and IgA titres (586EU median, p=0.0054) than normal subjects (320 and 28 median for IgG and IgA respectively) and a trend towards lower IgM titres (206EU median compared with 537EU, p=0.1029) than normal. IgG avidities tended to be higher (1.74M mean, p=0.065) in periodontitis than in normal subjects (1.21M mean). NAL subjects had higher mean IgM avidity (0.97M, p=0.0005) than AL subjects (0.70M). This study suggests that avidity of serum antibody to *P. gingivalis*, particularly the IgM subclass, may have prognostic significance in periodontal disease.

1078 Antibody Induction Against Treponema Antigens in Rapidly Progressive Periodontitis. P. PURUCKER*, R. LANGE, V. WOLF, H. W. KÖLMEL and J.-P. BERNIMOULIN (Free University and Humboldt University, Berlin, FRG):

Previous studies have shown, that T. denticola is involved in the pathogenesis of periodontitis. During active periodontal disease an IgG antibody induction against T. denticola is observed. It is unknown which cell structures are immunogenic. The aim of this investigation was to evaluate the prevalence of antibodies against different antigens of T. denticola. Untreated patients with rapidly progressive periodontitis (n=15), two patients groups with either Lyme borreliosis (n=15) or syphilis (n=15), and healthy control subjects (n=15) took part in the study. The peripheral blood IgG antibodies against whole spirochete cells, outer sheath, flagella, and protoplasmic cylinder were determined. For this analysis enzyme-linked-immunosorbent-assay (ELISA) and Western blot technique was used. To evaluate the specificity of the observed antibodies Treponema pallidum and Borrelia burgdorferi were used as control antigens. With T. denticola antigen there was no homogenous reaction pattern. B. burgdorferi antigen revealed no reactivity in periodontitis patients. In contrast to these findings periodontitis sera reacted with two antigen bands (14 kD and 50 kD) of T. pallidum. Out of this data we conclude, that patients with untreated rapidly progressive periodontitis have elevated antibodies against Treponema species. It is unclear wether this Treponema specific antibody production represents an immunresponse to a T. pallidum related species or another undetected spirochete. This study was supported by Berlin-Forschung and FNK Grant.

1079 Immunological analysis of Treponema denticola antigens. R. LANGE*, V. WOLF, P. PURUCKER, H.W. KÖLMEL and J.-P. BERNIMOULIN (Free University and Humboldt University, Berlin, FRG):

Treponema denticola is the most prevalent spirochete species in advanced periodontitis. There has been limited information about T. denticola at the immunological level. Therefore we cultured the T. denticola reference strain (ATCC 33521) and 4 isolated spirochetal strain from deep periodontal pockets. Polyclonal rabbit antisera were produced to the following T. denticola antigens: whole spirochete cells, outer sheath, flagella, and protoplasmic cylinder. As specificity control of the induced antibodies Treponema pallidum and Borrelia burgdorferi were used. Tricine-SDS-PAGE and immunoblotting were performed to analyse a possible variation of separated polypeptides. The results show, that most antigens seen on stained SDS-PAGE antigen profile are immunogenic. Two major antigen bands (12 kD and 66 kD) could be induced by preparation of outer sheath proteins. The analysis of the protoplasmic cylinder serum demonstrated a strong reactivity against an approximately 25 kD protein. The antiserum raised against the flagella preparation revealed, that the motility protein consists of two subunits with a molecular weight of 35 and 38 kD. Antibodies directed to this flagellin subunits reacted with the 38 kD but not with the 37 kD flagellin subunit of T. pallidum. No reaction with the 41 kD flagellin of B. burgdorferi was observed. The conclusion out of these findings is, that there are T. denticola specific antibodies present in preimmunized rabbit sera, which do not crossreact with other pathogenic spirochetes. This study was supported by Berlin-Forschung and FNK Grant.

1080 Characterisation of a monoclonal antibody to Porphyromonas gingivalis. V BOOTH*, PP ASHLEY, R GMUR and T LEHNER. (UMDS, Guy's Hosp, Univ London & Dental Inst, Univ Zurich)

P gingivalis has been implicated in the pathogenesis of periodontitis and monoclonal antibodies (Mabs) have been raised to P gingivalis. The Mab hybridoma 61BG 1.3 produces IgG1 antibodies. The aim of the present work were to investigate the specificity of this Mab to a variety of strains of P gingivalis and to identify its antigenic determinant. Mab 61BG recognised 21 laboratory strains of P gingivalis by immunofluorescence including representatives of the 4 serotypes. It also recognised all of the 109 isolates of P gingivalis cultured from human plaque. Western blotting of P gingivalis strains NCTC 11834 and W50 showed two major bands with Mr 42K and 31K. A third major band with Mr 39K or 36K was observed in the 11834 and W50 strains respectively. Both strains also exhibited multiple minor bands at higher Mr. Fimbrial extract, LPS and capsular material from P gingivalis were examined in order to identify the antigen recognised by the Mab. Only the capsular material reacted with the Mab in both Western blots and solid phase radioimmunoassays, suggesting that this Mab recognises a determinant in the capsule of P gingivalis. In conclusion, Mab 61BG reacted with all strains of P gingivalis tested. The results suggest that Mab 61BG recognises a capsular determinant.

1081 Serum antibodies to six species of *Selenomonas* in human periodontal disease. C.-H. Lai*, M.A. Listgarten, L.-T. Lai, T. Suwa and Y. Chan. University of Pennsylvania, School of Dental Medicine, Philadelphia, PA.

Selenomonas (*S.*) are anaerobic, gram-negative curved rods with tufts of flagella on the concave side of the cell. They are isolated from gingival crevices with gingivitis or periodontitis (Moore *et al., Int. J. Systematic Bact.*, 36:271-280, 1987). 25 patients with untreated adult periodontitis (AP), 25 with gingivitis (G) and 25 periodontally healthy subjects (H) were compared for serum antibody titers to *S. flueggei* (*Sf*), *S. infelix* (*Si*), *S. artemidis* (*Sa*), *S. dianae* (*Sd*), *S. noxia* (*Sn*) and *S. sputigena* (*Ss*). Serum IgA and IgG titers to the six species of *Selenomonas* were determined by an enzyme-linked immunosorbent assay (ELISA). Results of the ELISA were expressed as ELISA units (EU) and analyzed by a one-way ANOVA and a multiple comparison test. IgA antibody titers to *Sf, Si, Sn, Ss and Sd* were significantly elevated ($p<0.05$) in AP patients (*Sf*: 48 EU, *Si*: 90 EU, *Sn*: 127 EU, *Ss*: 46 EU, and *Sd*: 88 EU), as compared to group H subjects (*Sf*: 33 EU, *Si*: 62 EU, *Sn*: 84 EU, *Ss*: 32 EU, *Sd*: 42 EU). No statistically significant differences were found for *Ss* IgA antibody titers between the AP and H groups, or between titers to all *Selenomonas* species between the AP and G groups and, with the exception of *Sd*, between the G and H groups. IgA antibody titers to *Sd* were statistically significantly higher in the G than H group (59 vs. 42 EU, $p<0.05$). No statistically significant differences in IgG antibody titers to all species of *Selenomonas* were demonstrable among the 3 groups. These results complement our earlier findings of *Selenomonas* cells within the gingival tissues of patients with juvenile and adult periodontitis (Lai *et al. J. Dent. Res.*, 68:363, 1989, Abstract No. 1454). The presence of *Selenomonas* cells in the gingival tissues may result in the stimulation of an IgA, but not an IgG humoral immune response.
(Supported in part by NIH grant RO1-DE08085 and NIH grant RR-01224/00040).

1082 Longitudinal Antibody Responses to *A. actinomycetamcomitans* Outer Membrane Antigens. MJ STEFFEN* and JL EBERSOLE, Univ. of Texas Health Science Center, San Antonio, TX, USA

Aa positive (serological and microbiological) periodontal disease patients (11 adult (AP), 7 localized juvenile (LJP)) were examined for the presence of specific antibody responses to sarcosyl insoluble antigens of *Aa* strain Y4 outer envelopes (OMA). For each patient, we obtained from 6-13 samples over a period of 18-42 months during disease activity. Sera from these patients detected 22 different antigens in the OMA ranging in molecular weight from 14-97 kDa. LJP and AP responded to a mean of 35% and 41% of the 22 antigens, respectively. 2/9 sera from healthy subjects detected no antigens and 7/9 sera detected a mean of 7% of the 22 OMA ($p<0.0001$ versus LJP and AP). Longitudinally, 17/22 of the antigens could be detected in every sample from at least one patient and 5 antigens appeared to react variably among the patients sera. 17, 28, and 38 kDa antigens were commonly detected by diseased patients (>90%). Longitudinal samples from seropositive LJP and AP reacted 80-100% of the time to these antigens, in contrast to normal sera: 17 kDa (78%, $p=$NS); 28 kDa (0%, $p=0.0009$); and 38 kDa (22%, $p=0.0203$). Also, 15, 58, 65, 79 and 97 kDa antigens to which sera displayed a longitudinal response were noted. These antigens were detected 100% of the time in 33-83% of the seropositive patients. The 65 kDa antigen showed an increased frequency of reaction in the AP versus the LJP ($p=0.0104$). We conclude that: (1) the 17 kDa antigen is probably a cross-reacting antigen among various bacteria; (2) responses to the 28 and 38 kDa antigens are unique and may be important in the humoral response to *Aa* in periodontal disease; (3) response to the 65 kDa antigen exemplifies potential distinctions in responses to *Aa* between the two diseases; and, (4) longitudinal alterations in antigens present on a laboratory strain of *Aa* are not clearly descriptive of disease active episodes. Supported by DE-07809.